



STATEMENT OF BERTRAM W. CARNOW, M.D.

My name is Bertram Warren Carnow. I am a physician actively engaged in the practice of Occupational and Environmental Medicine, in the disciplines of Internal Medicine, Preventive Medicine, Toxicology and Epidemiology, in the areas of research, teaching and clinical medicine. I have developed and served as director of the Environmental Health Resource Center of the State of Illinois which functioned first, as a section in the Department of Preventive Medicine in the College of Medicine and then, as a component of the Department of Occupational and Environmental Medicine at the University of Illinois, School of Public Health. I also developed and chaired the Department of Occupational and Environmental Medicine at the University School of Public Health from its inception in 1972 until 1978. In 1976, I also became director of the Division of Occupational Medicine at Cook County Hospital.

I am currently professor of Occupational and Environmental Health Sciences in the School of Public Health and professor of Preventive Medicine in the College of Medicine, as well as director of the Great Lakes Center for Occupational Safety and Health, a federally funded center for the training of physicians as specialists in occupational medicine, and nurses in the College of Nursing, industrial hygienists in the School of Public Health, and safety engineers in the College of Engineering, in the disciplines of occupational and environmental health and safety. I am also President and Senior Scientist with

Carnow, Conibear & Associates, Ltd., an occupational and environmental health and safety consulting organization in Chicago, Illinois.

In pursuing my career in Environmental and Occupational Medicine, I have directed and carried out multiple studies of the effects of air and water pollutants on health. These have included studies on the effects of sulphur dioxide and particulate on the respiratory tract and the heart, and multiple studies examining the impact of environmental contaminants on individuals and populations at high risk because of age, socioeconomic factors, underlying disease or other factors which put them at excessive risk when exposed to environmental toxic agents.

Two areas of activities in which I have been engaged over the past number of years are particularly relevant to the problem to be discussed, namely, the impact of water contaminated by the polycyclic aromatic hydrocarbons and other chemicals on the health of a community. I was principal investigator of a federally funded study given in order to examine the health effects of barium in water. The barium water standard is, to some degree, based on the results of this epidemiologic study. I also sat as a member of the National Academy of Sciences panel on polycyclic organic matter, a toxic substance of major concern here. The results of multiple studies carried out by Professor Paul Meier and myself, at the University of Chicago, served as the basis for much of the conclusions arrived at by the panel

regarding the toxicity and, particularly, the carcinogenic potential of these polycyclic aromatic compounds.

I served as a member or chairman of a number of other panels on the effects of environmental pollutants on health for the National Academy of Sciences and was a member of its Medical and Biological Environmental Effects Committee for a number of years. I also served as a consultant on environmental cancer for the Environmental Protection Agency for a number of years, and on the effects of environmental pollutants on health for the Council on Environmental Quality of the President. My curriculum vitae is attached and presents my other professional activities.

I have been asked by the United States Department of Justice, the United States Environmental Protection Agency, and the office of the Attorney General of the State of Minnesota, to examine collected data which reveal that there has been a loss of containment of chemicals into the soil and water of the surrounding areas, including a number of wells in the St. Louis Park area. These chemicals were components of a coal tar product which was produced and used in industrial processes, and include polycyclic aromatic hydrocarbons, biphenyls, dibenzofurans, phenolic compounds and metals including chromium, manganese, lead, arsenic and zinc. I have also been asked whether, upon examining this information, I am able to render an opinion regarding the actual or potential health impact on the communities surrounding the Reilly Tar property and other communities which may

now, or in the future, be exposed to these contaminants in the soil, air and water. I have examined the data and do have an opinion. It is my opinion that the current loss of containment of these compounds into the air, water, and soil constitutes a clear and present danger to the health of individuals who may currently be exposed and who may have been exposed in the past. One study, which we have examined, the St. Louis Park breast cancer study (critique appended) appears to indicate that it may already have had negative health impact on at least one community. It is also my opinion that continued exposure to levels of the compounds known to be present in the environment, will result in damage to the health of many others. The balance of this document provides the basis for this opinion.

#### TOXICITY OF THE CHEMICALS

In attempting to make a determination of whether or not chemicals in the environment are a hazard to those who may come in contact with them, a number of toxicologic aspects must be considered. The first includes the inherent toxicity of these agents. Creosote, which was produced for over 50 years, was one of the main products produced by Reilly Tar at its St. Louis Park tar distillation plant. It is a heavy, oily, liquid made by the distillation of coal tar or wood, at temperatures above 200 degrees centigrade (Parmeggiani, 1983). Creosote contains mixtures of at least 200 major chemicals and thousands of other chemicals in small concentrations. Some of the most important include phenols, creosols, pyridines and many polyaromatic and heterocyclic compounds, the PAHs.

Creosote, by itself in high concentrations, can be acutely toxic to individuals who ingest it or whose skin and mucus membranes are exposed to it. It is a severe irritant to these organs and tissues, and depending on the duration and intensity of exposure, can cause a variety of lesions. Individuals with very sensitive skins including those with chronic skin disease, infants, and those prone to a hypersensitive response to irritants may be at risk even at low doses.

The major hazard of exposure to creosote and its derivatives, relates to its carcinogenic properties. These have been known for almost a hundred years. In 1898, Mackenzie reported a case of scrotal tumors in a creosote worker. In 1924, Cookson reported a case of epithelioma of the skin on the hand of a worker who had worked for many years in a creosote factory. This individual died even though there was amputation of the arm above the elbow, from the spread of the cancer to multiple organ systems. In 1956, Lenson reported a case of five primary cutaneous cancers on the face of a 64 year old man, who had worked in a shipyard painting creosote on planks for only three years, from 1947 to 1950.

In animals, Sall and Shear demonstrated the growth of skin epitheliomas in mice painted with creosote oil in benzene. Others including Poel and Kammer produced malignant papillomas by applying the distillates from by-products of coke ovens to the shaved skin of mice. They tested the pitch residue from fractional distillation and found that it had carcinogenic activity. Two grades of creosote oil

and a "crystal free" anthracene oil, widely used as a wood preservative were found to be actively carcinogenic in animals in 1957 by Poel and Kammer. Lijinsky, analyzing creosote to determine its carcinogenic constituents, identified benz(a)anthracene as a major carcinogenic component of creosote oil in 1957. Many other studies since the early 1900s found that painting of coal tar and other applications of it to animals would cause cancers of various organs.

While all of the components of creosote are of concern, a number of them have shown themselves to be major causes of cancer. Many of the polycyclic aromatic hydrocarbons are carcinogenic although the ability to cause cancer may vary widely. Some of these, including benzo(a)pyrene, are highly carcinogenic and have been shown to cause cancer in humans who have been exposed to them, not only of the skin, but of multiple organ systems. It has been recognized since 1775 when Percival Potts discovered that chimney sweeps were at high risk to cancer of the scrotum and that washing away the soot containing these polycyclic compounds resulted in a lowering of the incidence of these cancers. In 1925, Kennaway made the observation that chimney sweeps not only contracted skin cancer, but also cancers of the respiratory and gastrointestinal tract above the stomach.

In 1936, Kuroda and Kawahata, found lung cancer in 12 of 15 cancer cases among employees of a coal gasification plant in Japan. The association with the gasification process was made because lung cancer

was a relatively rare occurrence in Japan at that time. This excess of lung cancer associated with working in coal gasification plants was later confirmed by a large study by Sir Richard Doll in the United Kingdom.

A major advance in our understanding of the toxicity and carcinogenic potential of the polycyclic aromatic hydrocarbons occurred when a number of American investigators carried out a series of epidemiologic studies of coke oven workers. Dr. William Lloyd found that the death rate from lung cancer was 10 times greater than normal for those people working five or more years on top of the coke ovens. Those working on the side of the ovens, as well as the top, but who had less than five years of topside experience, had a lung cancer death rate that was almost three times the expected rate. Finally, Lloyd found that the death rate from lung cancer was three and a half times the expected rate for people employed in coke oven plants for five or more years. He also observed that coke oven workers employed at non-oven work areas may have a higher gastrointestinal cancer death rate than the general population. In carrying out his studies, he took into consideration many other potentially confounding factors including cigarette smoking, race and age. While cigarette smoking demonstrated its known effect of causing lung cancer, Dr. Lloyd's study showed that the cancer-producing agents responsible for the excess lung cancer seen in top-side oven workers had an effect independent of and much stronger than that of heavy cigarette smoking.

In addition to the excessive rates for lung cancer found by Lloyd in coke oven workers, Redmond, in a series of studies, observed a remarkable increase in the kidney cancer death rate in coke oven workers. The rate was seven and a half times greater than expected. These results were in agreement with a 1951 report with the British Registrar General which showed an excess of bladder and kidney cancer for men employed as "laborers and unskilled workers in coke ovens and gas works".

Of 70 polycyclic aromatic hydrocarbons including benzo(a)pyrene which were tested for their mutagenic, and therefore, carcinogenic potential by a screening test, the Ames test, 34 were found to be positive. Thus, many PAH in addition to benzo(a)pyrene may be responsible for the excess cancers observed in certain populations.

The argument that the levels of benzo(a)pyrene or any other carcinogenic PAH that may be present are very low, and therefore, of no consequence, is specious and dangerous for a number of reasons. In the first place, it is generally accepted by scientists in the field that, theoretically, there is no safe level for carcinogens. This means that at any level of a carcinogen it is possible to incur a change in DNA which may result in the development of an abnormal cell

line which may become malignant. For this reason, any level which is accepted as an environmental standard, represents a socially acceptable risk, which is to say that as a result of an exposure to carcinogens at that particular level there will be a small excess of cancers as a result of an exposure to carcinogens at that particular level. In this situation, even such a level is difficult, if not impossible to determine for a number of reasons. These include:

#### ADDITIVE AND SYNERGISTIC EFFECTS

Multiple compounds are acting additively or synergistically. These include many other polycyclic aromatic hydrocarbons besides benzo(a) pyrene which are called initiators and may in some cases, also be promoters. A cancer initiator is a compound which causes an alteration in the template of the cell, that is, in the DNA; and the cell may then lie dormant for some time until it is "turned on" by what is known as a promoter. The polycyclic compounds may be both, but many are generally strong initiators.

In addition to all of the polycyclics which may be taken into the body, there are other compounds which may be additive or synergistic with them. The dibenzofurans, like the dioxins, are extremely potent cancer promoters and the presence of dibenzofurans and the polycyclic aromatic hydrocarbons represents a remarkable increase in the potential threat to human health and particularly the threat of cancer. As previously mentioned, Sall and Shear observed a tumor-promoting effect for the basic fraction of creosote oil when this

fraction was applied to the skin of mice. In 1955 Boutwell and Bosch studied the capacity of phenol and more than 50 chemicals structurally related to phenol to promote malignant tumors in mice after a single application of dimethylbenzanthracene (DMBA). Treatment by DMBA followed by 13 weeks of phenol application to the skin induced tumors in 95 percent of the mice. In contrast, only 3 of 23 mice developed tumors 24 weeks after a single application of DMBA and no applications of phenol. Boutwell and Bosch also observed that dimethylphenols were as potent promoters of tumors as phenol itself. In 1958, Roe et al observed that creosote applied to the skin of mice resulted in incidences of both skin and lung tumors which were higher than the incidences found in untreated control mice. In 1967, Tye et al observed tumor-promoting effects of coal tar phenols in mice that had inhaled mixtures of phenols for 2 hours per day, 3 times weekly, for 55 weeks.

It is known that benzo(a)pyrene and some of the other polycyclic aromatic hydrocarbons are not the proximate carcinogens. This means that in their unchanged form they are not strong cancer producers. When they are metabolized or broken down, however, they form metabolites which are very active compounds and are highly carcinogenic. While this phenomena is not true of all of the PAHs, it is true of benzo(a)pyrene and some of the others.

The enzymes which break these compounds down into potent carcinogens are a group called the mixed function oxidases. These are produced on

demand by the body and the ability to produce them is genetically determined. However compounds such as the dibenzofurans, for example, may inappropriately increase the production of the mixed function oxidases by thousands to hundreds of thousands of times. By this mechanism, the dibenzofurans act as cancer promoters, in that they accelerate and intensify remarkably the cancer-producing ability of other compounds like benzo(a)pyrene and other PAHs. For example, 3-methylcholanthrene, a compound very similar in its actions to dibenzofurans, has been used by cancer researchers for many years to increase the yield of cancers in experimental animals when another compound is being tested for carcinogenicity.

In addition other carcinogens, for example arsenic, appear to be present at the Reilly Tar Site. Finally, others like lead may be co-carcinogens and thus may enhance the carcinogenic potential of the mix. There is thus present a dangerous combination of initiators, promoters and co-carcinogens all of them at a low dose, possibly, but each of them contributing. Many of the standards which are used in assessing acceptable levels of carcinogens rely on tests which examined those carcinogens as though no others were present in the environment. Further, some of these are cumulative in the body and are not easily or rapidly destroyed, so that the absorption into the body day after day and (possibly over a lifetime) remarkably enhances the effect of each and, in sum, the effect of all.

### MULTIPLE ROUTES OF ENTRY

Another factor which enhances the possibility that increased cancer will result from ongoing exposure relates to multiple portals of entry. Some of these components have been found in a number of wells and in the water supply used by people in the communities. Other contaminants have entered or threatened to enter surface water bodies which are used by many people for wading, swimming and boating. Also, fish in these waters and animals drinking from them may be contaminated and, if eaten, toxic agents such as the metals may be absorbed. The probability therefore exists that some, or many of these PAHs, metals and others may enter the body through skin absorption, inhalation and ingestion, thus multiplying the amount present.

### MULTIPLE SOURCES

In addition, there are other sources of these contaminants, so that these also will add to the body burden. Polycyclic aromatic hydrocarbons are found in tobacco smoke and are absorbed by smokers and those who are in the room with smokers. Diesel engines contain polycyclic aromatic hydrocarbons, as do other engines that burn fossil fuels, presenting an additional burden with increased risk of altering DNA in cells and producing cancer. A wide range of studies were carried out by Dr. Paul Meier and myself that encompassed the populations in the 48 continental states, 21 countries, in addition to migrant populations. These studies showed that benzo(a)pyrene, used as an index of air pollution, was significantly correlated with

variations in lung cancer deaths. Our studies, later published by the National Academy of Sciences as part of the polycyclic organic matter document, revealed that for every microgram of benzo(a)pyrene per thousand micrograms of air, there appears to be a 3-5 percent increase in deaths from lung cancer. Absorption therefore, of the contaminants emanating from Reilly Tar by persons living in the surrounding communities, would be added to similar toxic agents from other sources that are absorbed through inhalation, ingestion and, possibly, skin absorption.

#### LONG LATENCY AND IRREVERSIBILITY

Some of the problems relating to environmental cancer have been presented, namely the multiple compounds which may cause these diseases and their relationships to each other. An additional concern which must be considered is that cancer has a long latency period and when it appears, it is permanent, irreversible and often fatal. The need for preventive intervention before this level of response occurs is considerable.

Two other types of abnormalities which may result from exposure to some of these agents include mutagenicity and teratogenicity. The former relates to genetic alterations in the germ plasm which results in transmission of abnormalities from parents to their children. Some compounds, for example, perylene, cyclopenta(c,d)pyrene and fluoranthene exhibit greater mutagenic potencies than benzo(a)pyrene. Many of these compounds have been identified in the drinking water of

St. Louis Park. Thus, in addition to cancer, other irreversible changes may result from exposure over a prolonged period of time to these chemicals. Genetic alterations in both male and female may be transmitted to the next and subsequent generations. An increase in the rate of spontaneous abortions may also result from increased mutations since many of the changes are too damaging to allow the embryo to develop fully.

A number of these compounds including the dibenzofurans are teratogenic. This means they cause defects in the newborn as a result of contact with toxins in the body of the mother which cross the placenta while she is pregnant. While some birth defects may not be destructive and disabling, many are, and they represent a great cost to both the families and community where they occur.

Many of the chemicals cause diseases other than cancer of multiple organ systems including the central nervous system, the brain, the peripheral nervous system, the liver and the kidneys. These include the dibenzofurans, some biphenyls, phenols, lead, arsenic, manganese and possibly others. As with cancer, the abnormalities may be undetected and slowly progressive. When they emerge clinically they are also permanent and irreversible.

#### POPULATIONS AT HIGH RISK

Some segments of the population (and they may include large numbers of individuals) are at particularly high risk when exposed to these

compounds. Therefore, so-called acceptable levels do not apply to these individuals since they have been derived from studies of populations not at high risk. High risk populations include at least the pregnant woman, the unborn child, the very young, those with chronic disease, particularly renal disease, others with immune system dysfunction and those who have high mixed function oxidase inducibility.

The fetus may be at high risk because, in addition to the teratogenic and mutagenic effects that can occur as a result of exposure, there are also fetotoxic effects. That is, the possibility that the unborn child may develop diseases or cancer before it is born. The fetus is exposed to most materials present in the maternal bloodstream. The fetus has underdeveloped metabolic systems for destroying the toxic materials and is more susceptible to damage because its organ systems are rapidly developing.

Young people are at high risk because of the latency period related to cancer. It may take 15 to 20 years before the cancer manifests itself, although in young people, certain types of malignancies (for example leukemia) may appear very early. The young are also at high risk because they tend to play in the dirt and in areas where surface water is present. Because of the increased metabolic activity of their bodies they may respond to a much greater degree to smaller doses of toxic agents. It has been shown, for example, that even relatively low levels of lead entering the body over a period of time

may cause mental retardation in children. The body can remove 300 micrograms of lead per day, but given multiple sources which provide more than this each day, there may be an accumulation of it in the body. This is also true of arsenic and may be true of other compounds to which these individuals may be exposed.

The aged and others with chronic diseases are also at high risk. Hypertension as a result of renal disease, or idiopathic hypertension which ultimately results in renal disease, may cause the body to retain many of these compounds longer than they otherwise might, since many of them exit through the kidneys. Accumulation thus is enhanced and the potential for increased effects is present.

#### SUMMARY AND CONCLUSIONS

1. A large number of chemicals and compounds are contained in creosote, a product produced by Reilly Tar over a 50 year period with loss of containment of many of them into the surrounding environments.
2. These compounds have been found in wells, creeks and soils away from the original site where they were produced.
3. Many of these compounds individually and in sum are carcinogens and have other toxic properties which may seriously affect human health including mutagenicity, fetotoxicity, and teratogenicity. In addition, they may

affect other organs including the central and peripheral nervous system, the liver, the skin, and possibly other organ systems.

4. In addition to the multiplicity of compounds which are carcinogenic, and which may be additive or multiplicative in their effects, there is the potential for synergistic action of some of them, particularly the cancer initiators, (the polycyclic aromatic hydrocarbons) and the promoters (the dibenzofurans and possibly the biphenyls). This may remarkably enhance the effect of each.
5. While levels of each of these contaminants may be small, they are dispersed throughout the environment where people live and may be absorbed on a daily basis through drinking, bathing and inhalation of dust and vapors. Their levels may build up in the body and for this reason they constitute a serious health hazard.
6. The diseases caused by these compounds, particularly cancer, are uniquely dangerous. They have long latency periods and may appear 10 to 30 years after initial exposure. Once they appear, they are permanent and irreversible and frequently fatal. It is therefore unreasonable to wait until symptoms of disease or increased disease rates

develop. Prudence dictates that measures be taken long before the onset of disease to prevent their occurrence.

It is for all of these reasons that I express the opinion that every attempt must be made to contain these toxic agents, remove them from the environment, and in the interim, assure that individuals who may be potentially exposed will be protected against the possibility of absorbing and retaining the toxic chemicals in their bodies.

## ADDENDUM

### Rebuttal and Critique

Arguments have been raised by Reilly Tar by implying the existence of safe threshold levels for carcinogens and that persons are exposed to carcinogens every day and that the human body has defense mechanisms or processes that detoxify chemicals or repair damage done by carcinogens. (Poel, 1980 pp. 55, 59). While some scientists believe that repair processes most probably exist at the molecular level (i.e. DNA level), no scientist has demonstrated their existence. Furthermore, the body does try to detoxify carcinogens just as it does chemicals that are toxic but not carcinogenic. However, for many carcinogens, the detoxifying mechanisms actually produce materials (i.e. metabolites) that are much more carcinogenic than the original carcinogen. This is especially true for PAHs (IARC, 32, 1983, p. 57). The high interest in hydrocarbon metabolism was precisely because PAHs, like many environmental carcinogens, were known to be unreactive, and that their adverse effects had to be explained by the by-products produced when body processes tried to detoxify them (IARC, 32, 1983, p. 57). Some PAHs stimulate their own metabolism by inducing mixed function oxidase activities in body organs and especially in the liver. Attention has been focused on one of these activities, that of aryl hydrocarbon hydroxylase (AHH). The inducibility of AHH is genetically determined. Those individuals who have increased AHH inducibility are at a greater risk to PAH-induced cancers. The continuous exposure to the carcinogenic PAHs found in the potable water supply of St. Louis Park means that individuals are

receiving additional doses of carcinogens besides the ones they are receiving from other sources. If the carcinogens were removed from these water supplies, their risk of contracting cancers would be reduced.

The arguments of Reilly Tar are based, in part, on a theory proposed by Jerina in 1977 (Jerina, et al., 1977; IARC #32, 1983, p. 61). The theory states that those benzo-ring diol epoxides in which the epoxide is in the bay region of a hydrocarbon molecule should display high biological activity. The International Agency for Research on Cancer (IARC) reports that studies indicate that bay-region diol epoxides are ultimate carcinogenic metabolites in about a dozen PAH. The IARC, however, does state, "It should be emphasized that the demonstration of high biological activity of a metabolically formed bay-region diol epoxide does not preclude the existence of other ultimate carcinogenic metabolites of PAHs, and that not all carcinogenic hydrocarbons have formal bay regions." (Ibid., p. 61).

Reilly Tar argues that this "theory has been confirmed by numerous experiments with PAH" and that the "potential carcinogenicity of PAH is dependent on their ability to be converted by metabolism in the body to a specific reactive intermediate most probably a diol-epoxide. Therefore, the carcinogenic activity of an individual PAH is dependent upon the extent to which these diol-epoxides are formed from the parent hydrocarbon." (Andelman and Santodonato, p. I-12). This statement is scientifically untrue and misleading. The metabolism of

PAH is not limited to the production of diol-epoxides and some carcinogenic PAHs do not metabolize to diol-epoxides, and thus, their carcinogenic properties cannot be explained by the presence of diol-epoxides.

The study of structure-activity relationships make up a small but important part of the assessment of a chemical for potential carcinogenic properties. Short-term screening tests in bacteria and mammalian cell lines are also used in such assessments. Comparison of the molecular structure of a chemical with the structures of known carcinogens is used to identify potential carcinogens and thereby supplement in vitro short-term tests for potential carcinogenicity. (Purchase, 1978. p. 904). The best known short-term screening test is the Salmonella/Ames test for mutagenicity. The Ames test predicts the carcinogenicity of a chemical with an accuracy of about 90%. Table 1 lists many PAHs that are found in the drinking water supply of St. Louis Park. The PAHs listed in the table are positive for mutagenicity in the Ames test. If the accuracy of the Ames test for predicting carcinogenicity holds, many of the PAHs found in the drinking water of St. Louis Park will ultimately be assessed as carcinogens. Some of these PAH include 1-methylnaphthalene, acenaphthylene, pyrene, benzo(e)pyrene, perylene, benzo(g, h, i) perylene, and fluoranthene. It should also be noted that the mutagenic potencies (as measured by the Ames test) exhibited by perylene and fluoranthene are greater than the potency of benzo(a)pyrene.

Comments on Minnesota Dept. of Health Epidemiological Study of Excess Breast Cancer in St. Louis Park

Due to the presence of polynuclear aromatic hydrocarbons (PAH) in the drinking water of the Minneapolis suburb of St. Louis Park, the Minnesota Department of Health performed a study in 1979/1980 to determine if there was excess cancer in this community. The paper reporting the results is entitled "Cancer Rates in a Community Exposed to Low Levels of Creosote Components in Municipal Water" (Appearing in the November, 1980 issue of Minnesota Medicine).

Site specific cancer rates were calculated for St. Louis Park, surrounding communities, and the Minneapolis-St. Paul Standard Metropolitan Statistical Area (SMSA). The rates were adjusted for age, sex, and race. The major finding was that females in St. Louis Park had a statistically significant excess of breast cancer compared to the other study groups. The excess was close to 50%. In addition, Edina, a neighboring community which was also thought to be contaminated, had a high rate of breast cancer, though not statistically significant. This study, although not conclusive, provides evidence of excess female breast cancer.

The method of analysis was the appropriate first step in determining if the water contamination was having an adverse health effect on the people of the St. Louis Park area. The results indicate that the presence of PAH's in the drinking water is having the expected effect on the population of St. Louis Park, namely, excess cancer (and, in

particular, breast cancer). The next step would be to determine if the excess breast cancer rate could be attributed to a higher prevalence in St. Louis Park of known risk factors for breast cancer, such as late age at first birth and family history of breast cancer. If this were the case, then it is possible that the contaminated water has had little effect on breast cancer rates.

The best epidemiological method to use in order to determine this is the case-control study. For this particular situation, what would be done would be to obtain as many breast cancer cases as possible from the St. Louis Park area and match them with controls, i.e. people from the St. Louis Park area who do not have cancer but are of the same age. The same would be done for the Minneapolis-St. Paul SMSA. After determining the prevalence of known risk factors for the different groups, one could determine for each population how much of the cancer could not be explained by the presence of known risk factors. If the rate of unexplained cancer were still significantly higher for the St. Louis Park area, then it would be even more likely that this was due to the PAH contaminated water.

As the Minnesota Department of Health indicate at the end of their paper, a case-control study was planned as the next step. The Minnesota Legislature has recently directed the Commissioner of Health to evaluate the feasibility of conducting further epidemiologic studies.

**TABLE I**  
**POLYCYCLIC AROMATIC HYDROCARBONS THAT TESTED POSITIVE**  
**IN THE AMES/SALMONELLA SHORT TERM SCREENING TEST FOR MUTAGENICITY<sup>1</sup>**

Acenaphthalene	Chrysene <sup>+</sup>	1-Methylpyrene
Acenaphthylene	*Cyclopenta(c,d)pyrene	1-Methylphenanthrene
Anthanthrene	Dibenz(a,c)anthracene <sup>+</sup>	2-Methylphenanthrene
Benz(a)anthracene <sup>+</sup>	Dibenz(a,h)anthracene <sup>+</sup>	4-Methylquinoline
7H-Benzo(c,d)anthracene-7-one	Dibenz(b,e)fluoranthene	*Perylene
Benzo(b)fluorene	7,12-Dimethylbenz(a)-anthracene <sup>+</sup>	4-Phenyl-pyridine
Benzo(a)pyrene <sup>+</sup>	m-Dinitrobenzene	Pyrene <sup>+</sup>
Benzo(e)pyrene	*Fluoranthene	Pyridine
Benzo(g,h,i,)perylene <sup>+</sup>	2-Methylanthracene	Quinoline
3,4-Benzoquinoline	9-Methylanthracene	Triphenylene
5,6-Benzoquinoline	3-Methylcholanthrene	
7,8-Benzoquinoline	1-Methylnaphthalene	

<sup>+</sup> Carcinogen

\* Mutagenic potency greater than that of benzo(a)pyrene.

<sup>1</sup> Kaden, DA et al (1979) Mutagenicity of soot and associated polycyclic aromatic hydrocarbons to salmonella typhimurium. Cancer Res 39:4152-4159.



Bertram W. Carnow, M.D.

3478

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Michael Reese Hospital  
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Basic Science Resident in Cardiology

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### **MILITARY SERVICE:**

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### **PROFESSIONAL EXPERIENCE:**

Director of Occupational and Environmental Medicine,  
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**OTHER APPOINTMENTS:**

Associate Editor, American Journal of Industrial Medicine

Member, Editorial Board, Journal of Safety Review

Member, Environmental Committee of the Chicago Institute of Medicine

Member, Epidemiology and Statistics Task Force  
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Member, American Heart Association, Epidemiology Committee

Member, American Heart Association, Task Force on Heart in Industry

Chairman, Committee for Review of NIOSH Criteria Documents,  
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Chairman, National Research Council, Panel on SO<sub>2</sub>,  
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Member, Panel on Non-Renewable Resources,  
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Member, Panel on Polycyclic Organic Matter,  
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Member, Medical and Biological Environmental Effects Committee,  
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Hearing Officer for the Council Environmental Quality  
National Hearings on ERDA Energy Programs

Reviewer, Extra-Mural Grants, Environmental Protection Agency

Chairman, APHA and Ford Foundation Task Force on Health  
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Air Pollution Technical Advisory Board, City of Chicago

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**CONSULTANT TO:**

Environmental Protection Agency Criteria Document on Carbon Monoxide

Environmental Protection Agency Criteria Document on Lead

National Academy of Sciences Study Team on Environmental Considerations Associated with Nonrenewable Materials

City of El Paso, Texas - Lead and SO<sub>2</sub> Pollutants

Minister of the Environment, City of Toronto, Province of Ontario, Canada on Lead Poisoning in Children

Environmental Protection Agency Office of Manpower Development

Senate Commerce Sub-Committee on Health Effects of Lead

U.S. Atomic Energy Commission on Health Effects of SO<sub>2</sub>  
(State of Pennsylvania Hearings)

National Institute of Environmental Health Science on SO<sub>2</sub>

Environmental and Occupational Consultant to the Government of the State of Illinois

American Public Health Association and the Academy of Pediatrics on Lead Poisoning in Children

Meat Packer's Union on PVC Asthma

AFL-CIO Industrial Union Department on Standards in the Workplace

Senate Sub-Committee on the Environment - Sulfates and Intermittent Stack Technology

President's Council on Environmental Quality

State of Illinois Institute of Environmental Quality

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State of California on the Health Effects of Lead

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State of Pennsylvania on the Health Effects of Coke Oven Operations

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Federal Occupational Safety and Health Administration on Standards and Enforcement and Health Education

Criteria Review Committee, National Institute of Occupational Safety and Health

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**PROFESSIONAL ORGANIZATION MEMBERSHIPS:**

American College of Preventive Medicine - Fellow

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American College of Toxicology - Fellow

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